



NPCR Education and Training Series (NETS)

Module10: Male Genitourinary Malignancies

Part 3 Testis

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Advanced Abstracting Testicular Cancer

I. GETTING READY TO ABSTRACT

Incidence, Work-up, Anatomy, Histology

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There's much about testicular cancer that can be confusing. We hope to dissipate that confusion in the next hour.


This section of advanced abstracting for testicular cancer discusses incidence, symptoms, diagnostic procedures, the anatomy of the testes and their regional lymph nodes, and the various histologic types of testicular cancers.

Cancer Facts

- ◆ **1% of all male cancers**
 - About 8,000 new cases per year
 - 380 deaths per year
- ◆ **Median age 34**
 - Most common cancer ages 15–34
- ◆ **Whites have 5 times incidence rate of blacks**
- ◆ **1–3% bilateral**
- ◆ **90+% curable, even in late stage**



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According to the American Cancer Society, there will be an estimated 7,920 new cases, and an estimated 380 deaths in 2008.

SEER reports that the median age of testicular patients is 34 years. It is the most common cancer among young males.

Race is an interesting factor. Caucasian males have 5 times the rate of testicular cancer than African American males, and twice the rate of Asian males. Hispanic males are somewhere between the 2 to 5 times rate. The reason for this is unknown.

1–3% of cases arise bilaterally in the testicles but, unlike ovaries, you would abstract each separately.

This cancer is highly curable, thanks to chemotherapy, which we will discuss later in the lecture.

The Orchid Cancer Appeal is the fundraising effort in England for testicular and prostate cancer. Orchid and orchiectomy have the same root word in Greek, orchis, Greek for testicle.

Age-Adjusted Incidence Rates by Race For Testis Cancer, All Ages, Males 1975–2004

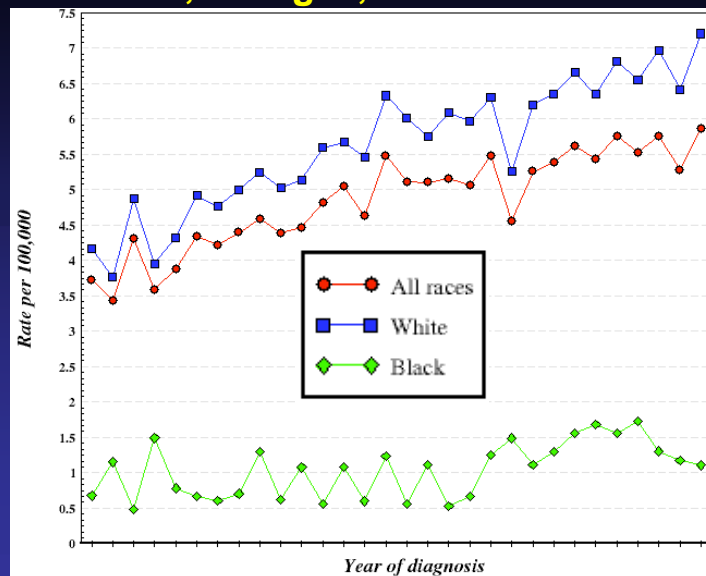


Image Source: National Cancer Institute

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SEER Age Adjusted Incidence Rates by Race For Testis Cancer, All Ages, Males

SEER 9 Registries for 1975–2003

Age-Adjusted to the 2000 U.S. Standard Population

(Rates are expressed as cases per 100,000).

The incidence of testicular cancer in the United States continues to climb, although there is no readily apparent reason.

Risk Factors

- ◆ Cryptorchidism (C62.0 vs C62.1)
- ◆ Congenital abnormalities
 - Testes, penis, or kidneys
 - Inguinal hernia
- ◆ History of testicular cancer
- ◆ Family history (father, brother)
- ◆ Genetics: TGCT1 found
- ◆ HIV/AIDS
- ◆ Body size
- ◆ Moles

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Undescended testicle (cryptorchidism): Normally, the testicles descend from inside the abdomen into the scrotum before birth. The risk of testicular cancer is increased in males with a testicle that does not move down into the scrotum (14% of cases). This risk decreases after surgery to move the testicle into the scrotum, but does not reach the risk level of a normally descended testicle. The increased risk applies to both testicles (25% of "normal" testicles with history of cryptorchidism in other testicle). Some doctors think it's not the cryptorchidism that raises the risk of cancer, but some other problem that causes the cryptorchidism and the cancer. The risk of developing testicular cancer has been estimated at 1 out of 20 for a testis retained in the abdomen, and 1 out of 80 if the testis was within the inguinal canal.

Congenital abnormalities: Men born with abnormalities of the testicles, penis, or kidneys, as well as those with inguinal hernia (hernia in the groin area, where the thigh meets the abdomen), may be at increased risk.

History of testicular cancer: Men who have had testicular cancer are at increased risk of developing cancer in the other testicle.

Family history of testicular cancer: The risk for testicular cancer is greater in men whose brother or father has had the disease. But it's still rare to find it occurring in families (per NCI website).

Genetics: The newly located gene, which has been called TGCT1, makes men who carry it more susceptible to testicular germ cell tumors (TGCT), which make up 95% of all testicular cancer cases. TGCT1 is inherited through the mother on chromosome X (per English research 1999).

HIV/AIDS: Men who have HIV, especially those that have developed full-blown AIDS, have increased risk. No other infection has shown a relationship to testicular cancer.

Body Size: Per a study done in Sweden, tall slim males may be at increased risk.

Moles: Males who have many moles or skin spots may be at increased risk. These moles are found on the back, chest, face, and belly; and are called multiple atypical nevi. Why this is true is not known. These patients might also have a greater risk of melanoma.

Screening

◆ Screening not recommended

- Good survival rate, even at later stage
- Not cost-effective

◆ Testicular Self-Exam

- After shower
- Roll both between fingers
- Check epididymis

Image Source: National Cancer Institute



Testicular Self-Exam (TSE)



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Screening is not recommended for two reasons. Many physicians do not feel it is necessary, since finding testicular cancer earlier would not change the mortality rate (which is already excellent). Secondly, it would not be cost-effective to have a formal screening program.

Young males should be taught to do a self-exam. It should be done after a shower or bath because the skin is more relaxed. Both testicles should be rolled between the fingers, checking for lumps. The epididymis should also be checked. There are information sheets and shower cards available from ACS or other support groups. Men should be reminded that one testicle is normally slightly larger than the other.

Symptoms

- ◆ Painless lump or swelling
- ◆ Pain or discomfort
- ◆ Enlargement, “funny” feeling
- ◆ Heaviness in scrotum
- ◆ Dull ache in back, groin, or abdomen
- ◆ Fluid collection in scrotum
- ◆ Enlargement/tenderness breasts

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The various symptoms listed on this slide are not necessarily 100% indicative of cancer. An infected or injured testicle can also cause these symptoms. But any of these symptoms is worth getting checked out.

References include American Cancer Society and <http://www.webmd.com>

Workup

- ◆ H&P
- ◆ Ultrasound
- ◆ Tumor markers
 - LDH, AFP, hCG
- ◆ Biopsy
- ◆ Chest X-ray and other radiology for staging (CT, MRI, PET)

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Physical exams should include a testicular exam, as well as palpation of abdomen for possible enlarged nodes.

A suspicious physical examination would lead to a sonogram or ultrasound to see if any mass is solid or cystic.

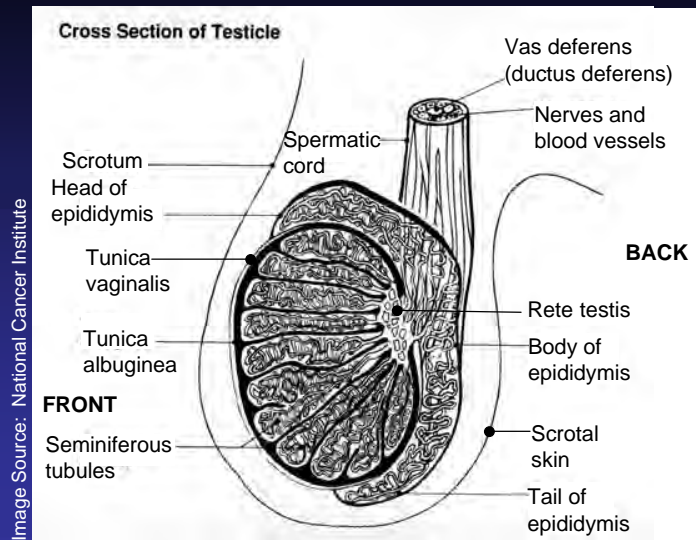
Tumor markers will be discussed later in the collaborative staging section. During a patient work-up, one of the uses of markers is to indicate what kind of testicular cancer may be present.

Biopsy is listed, but it is rare that a needle biopsy would be done of a suspicious lesion. Most suspicious lesions are resected.

Other radiology is done, especially of the nodal area and of the lung (which is usually the first distant metastatic site).

References: NCCN guidelines

Internal Testicular Anatomy



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The testicles (also called testes or gonads) are the male sex glands. They are located behind the penis in the scrotum. The testicles produce and store sperm, and they also serve as the body's main source of male hormone production. These hormones control the development of the reproductive organs and other male characteristics, such as body and facial hair, low voice, wide shoulders, and libido.

There are three ICD-O-3 primary site codes for testis: C62.0, undescended testis; C62.1, descended testis; and C62.9, testis, NOS. An undescended testis remains in the abdomen or inguinal canal. A descended testis (whether natural or surgically placed) is in the scrotum. If there is no statement whether the testis is in the abdomen, inguinal canal, or scrotum, the case may be coded as C62.9, testis, NOS. Code the location of the testis (undescended or descended) at the time of diagnosis.

Anatomy Vocabulary

- ◆ **Leydig cells**—secrete testosterone
- ◆ **Tunica albuginea**—dense capsule around
- ◆ **Tunica vaginalis**—serous covering
- ◆ **Rete testis**—network of efferent ducts
- ◆ **Epididymis**—storage vessel for sperm
- ◆ **Vas (ductus) deferens**—carries sperm to urethra

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Leydig cells form during the 16th and 20th week of gestation and are quiescent until puberty, when they begin secreting testosterone.

The tunica albuginea forms the capsule around the seminiferous vessels and the septa that divide the vessels into compartments called lobules. There is a tunica albuginea in the penis, ovary, and spleen as well as the testicle.

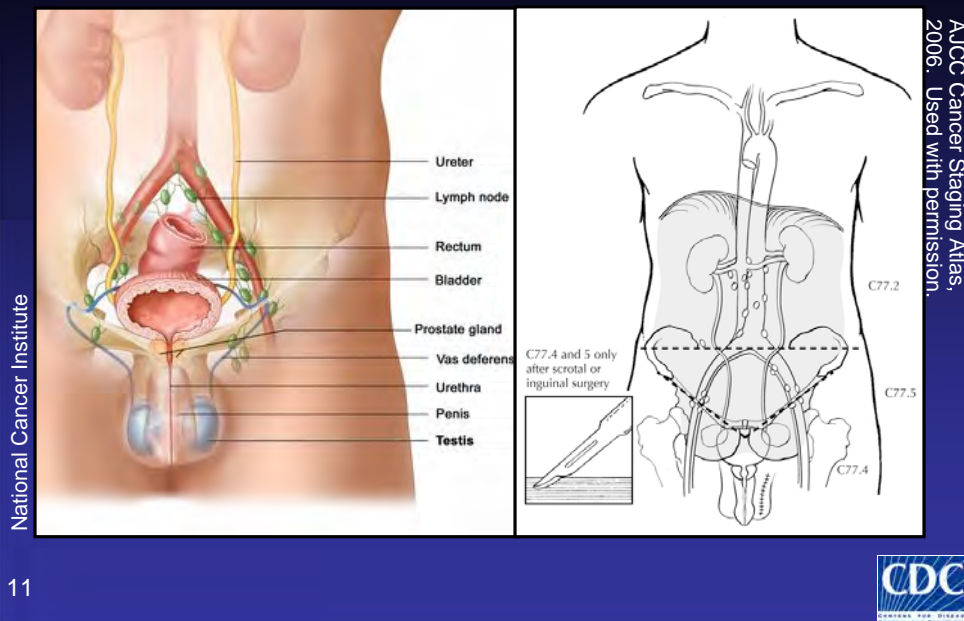
The tunica vaginalis is the serous covering of the front and sides of the testicle and epididymis. It is similar to the serosa on the GI organs. In the fetus, it came from the peritoneum. There are two layers to the tunica vaginalis, parietal and visceral. These are similar to lung.

The rete (ree-tee or rate) testis is similar to the renal pelvis in the kidney. It is the area where the seminiferous tubules of the testicle are concentrated.

The epididymis (epi-did'-i-mus) is a tightly coiled tube connecting the efferent ducts from the rear of each testicle to the vas deferens.

The vas deferens (ductus or “carrying away” duct) are muscular tubes (surrounded by smooth muscle) connecting the left and right epididymis to the ejaculatory ducts. These propel the sperm forward at the appropriate moment.

Lymph Nodes

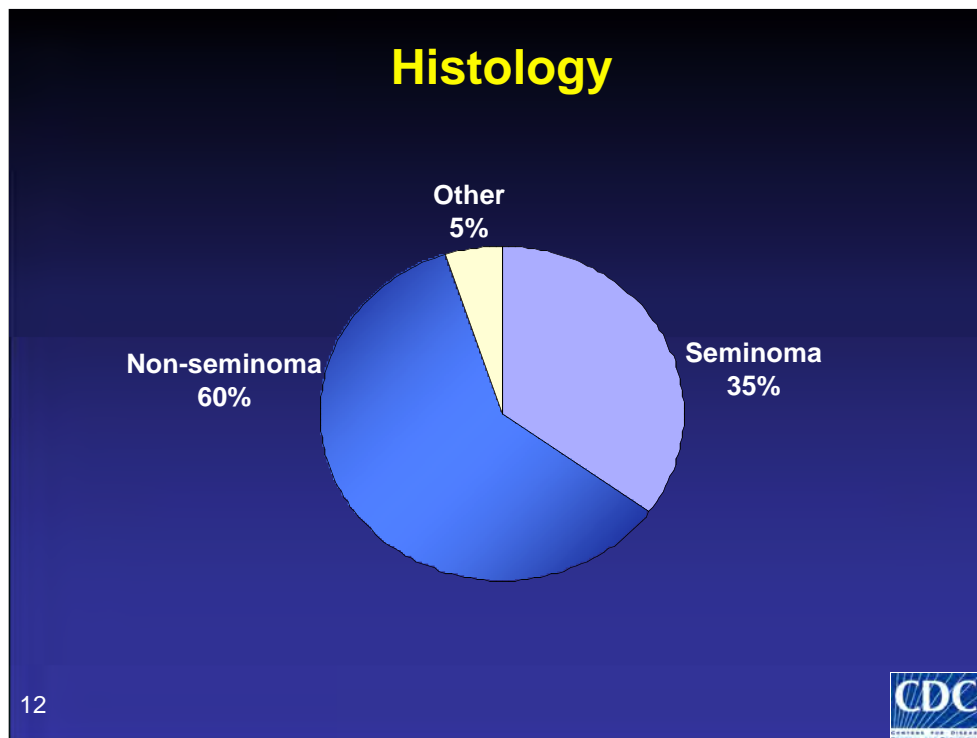


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The regional lymph nodes for the testes are shown in green on the left illustration.

There is something we need to understand about the relationship between the testicles and their regional lymph nodes. In the fetus, the testes develop in the region of the kidneys. They start to descend at the 12th week of gestation, reaching the internal orifices of the inguinal canal at mid-gestation. They move into the scrotum during the last two months of gestation. As the fetus is developing, the regional nodes are higher in the pelvis and they retain their “connection” when the testes descend into the scrotum.

Therefore, the regional lymph nodes of the testis are the retroperitoneal and intraabdominal lymph nodes (C77.2) at the level of the kidneys, including the interaortocaval, para-aortic, paracaval, preaortic, precaval, retroaortic, and retrocaval. The intrapelvic (C77.5), the external iliac, and inguinal nodes (C77.4) are considered regional only after scrotal or inguinal surgery; prior to diagnosis of the testicular tumor.

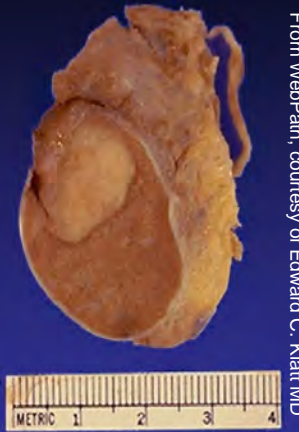


For the next few slides, we're going to discuss the types of histology seen in testicular cancer. Basically, we're looking at 30–40% seminomas (versus 60% non-seminomas), both of which may be referred to as germ cell tumors. There is also a small percentage of other histologies that can be included in the testicular cancer group.

Source: American Cancer Society

Seminoma

- ◆ 75% Stage I
- ◆ Classic – Age 40-59
 - Mets to LNs
 - Usually negative AFP
- ◆ Spermatic – Age 60-69
 - Indolent



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Seminomas are frequently found in stage I. They can be subdivided into classic and spermatic seminomas. Classic seminoma is most frequent in 40–59 year olds, although it might occur in younger patients. They might develop LN metastasis, but usually have a negative AFP tumor marker.

Spermatic seminomas occur in older patients, but are usually fairly indolent tumors.

How do we tell them apart? Registrars don't. It requires pathologic definitions of mitotic rates and cell appearance to give a specific designation. Per <http://pathologyoutlines.com>, anaplastic seminoma is an outdated term.

Resources: American Cancer Society, <http://pathologyoutlines.com>, Testicular Cancer Resource Center

Non-Seminomatous Types

◆ Embryonal

- 2nd most common
- Usually 2nd decade
- AFP and hCG +
- CEA neg
- 30% para-aortic LN, lung or liver mets at dx

◆ Yolk Sac

(Endodermal Sinus)

- Usually in children
- Frequently liver mets
- AFP+, hCG neg

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There is quite a variety of non-seminoma tumors. We will look at the pure types, meaning that is the only cell type present in the specimen.

Embryonal carcinoma is the second most common type of pure testicular tumor, although 85% of mixed tumors have an embryonal element to them. 65% have metastases at diagnosis. They are often associated with symptoms (back pain, dyspnea, neurologic symptoms). Treatment is controversial, as 97% of stage I cases are disease free after orchiectomy. Some physicians recommend watchful waiting, others recommend retroperitoneal lymph node dissection and chemotherapy if nodal metastases are present.

Yolk sac carcinoma is the most common testicular tumor in boys age 3 years or younger. It has a good prognosis at this age (80%+ are stage I). These are often pure with no other elements. If an adult has a yolk sac tumor, it is usually part of a mixed tumor and has a prognosis similar to embryonal carcinoma.

After treatment, this cell type may give rise to spindle cell sarcoma with myxoid or collagenous stroma later in life, and would be abstracted as a second primary.

Resources: American Cancer Society, <http://pathologyoutlines.com>, Testicular Cancer Resource Center

Non-Seminomatous Types

◆ Choriocarcinoma

- Frequent in mixed tumors
- AFP neg, hCG pos
- Poor prognosis
- May present with mets

◆ Teratoma

- Frequent in mixed tumors
- “Benign”
 - ◆ Young patients – mature tumors
 - ◆ Older patients – immature tumors
- Surgery is only effective treatment
- hCG, AFP not elevated

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Choriocarcinomas are identical tumors to those that arise in placenta, ovary, mediastinum, or abdomen. They come from sequestered cell rests. They are usually fatal if pure choriocarcinoma, but this cell type is frequently seen in mixed tumors. These tumors may present with metastases (lymph node, lung, or liver), but little or no lesion is seen in the testicle.

Mature teratomas have all three germ cell elements (endoderm, mesoderm, ectoderm), and frequently contain skin, bone, teeth, or hair. In females, these may be seen in the ovary.

Immature teratomas are more aggressive, and have tissues that are not recognized as normal elements.

Teratomas may undergo a malignant transformation.

AML occurs in patients with history of teratoma.

In children, usually age 3 or younger, teratoma is the second most common testicular tumor after yolk sac. It is not associated with intratubular germ cell neoplasia. There is no need for lymph node dissection after orchiectomy since teratomas almost never metastasize. Teratomas are usually pure; they have been associated with Down's syndrome, Klinefelter's syndrome, xeroderma pigmentosa, spina bifida, and hemihypertrophy.

Adult teratomas are rare, about 2–3% are pure. They can recur as teratoma (14%), or transform to embryonal carcinoma (18%) in metastasis.

Resources: American Cancer Society, <http://pathologyoutlines.com>, Testicular Cancer Resource Center

Other (Non-Germinoma)

- ◆ Sex cord stromal tumor
 - Leydig cell (/0, /1, or /3)
 - Sertoli cell (/0, /1, or /3)
 - Granulosa-theca cell (/0, /1, or /3)
- ◆ Gonadoblastoma (/1)
- ◆ Lymphomas and leukemias
- ◆ Rhabdomyosarcoma
- ◆ Others – rare

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The sex cord stromal tumors are similar to ovarian tumors, and have a similar appearance and presentation in male patients. These tumors are reportable when stated by the pathologist to be malignant, but they can also be benign or borderline tumors. You may need to consult with your pathologist to confirm reportability.

Leydig cell tumors are very strange. In children, they cause an increase in androgen production, leading to deep voice, hirsutism, precocious development, and sexual interest. But in adults, there is increased estrogen production (yes, in males), producing gynecomastia, female hair distribution, genital underdevelopment, and loss of libido.

Sertoli cell tumors are usually benign and may be bilateral or multifocal; but they may be found as malignant when they are usually solitary and unilateral.

There are two types of granulosa-theca cell tumors: adult (rare, non-functioning, and usually benign) or juvenile (most common neonatal testicular tumor).

Gonadoblastoma may be found in males with abnormal external genitalia (could be male, female, or ambiguous).

Lymphomas and leukemia may be found in the testicles. 50% of males with bilateral tumors have lymphoma, usually B cell type, but it could also be Hodgkin or Burkitt. Leukemic involvement of testis is common with ALL or AML, with bilateral involvement common.

The testis may be first site of relapse, especially in children.

Rhabdomyosarcoma may not be a testicular tumor, but a scrotal tumor that has invaded the testicle. Review these carefully with your pathologist, and code the primary appropriately to the true site of origin (most likely scrotum).

What else? Mesothelioma, plasmacytoma, PNETs... so many possibilities, but these are rarities.

Resources: American Cancer Society, <http://pathologyoutlines.com>, Testicular Cancer Resource Center

Extragenadal Germ Cell Tumors

- ◆ Germ cells outside the sex organs
- ◆ Usually mediastinal, retroperitoneal, or pineal
- ◆ May have carcinoma in situ in testicle if retroperitoneal

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In the fetus, germ cells lie outside the embryo in the yolk sac. They are supposed to migrate into the gonads in fetal development, but sometimes the cells wander elsewhere. If they become malignant later in life, they are referred to as extragonadal tumors. The variety of histologies, including benign, that we have seen in the testicle (or ovary) can also be found in these extragonadal tumors.

Extragenadal germ cell tumors usually develop in the chest, retroperitoneum, or pineal area of the brain, and if no testicular cancer is found, we code to the extratesticular site where the cancer was found. Treatment of these depends on where they are as well as what they are.

If the patient can be convinced to have an orchiectomy (usually due to suspicious sonogram or other evidence), carcinoma in situ may be found in the testes. If CIS is found in testes, physicians may suspect that the testicular cancer regressed within the testes, but only after it metastasized.

Resources: American Cancer Society, <http://pathologyoutlines.com>, Testicular Cancer Resource Center

Coding Mixed Tumors

◆ Germ cell in order of prognosis

- Choriocarcinoma 9100
- Yolk sac 9071
- Embryonal 9070
- Teratoma 9080
- Seminoma 9061-9064

◆ If one of the cell types is:

- Choriocarcinoma, use 9101
- Embryonal cell, use 9081
- Teratoma, use 9081
- Seminoma and non-seminoma, use 9085
- If no seminoma, use 9065

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This information is from the SEER Coding Guidelines 2002. Testicle was not included in the 2007 multiple primary and histology coding rule changes in the first round, although there may be changes within the next five years.

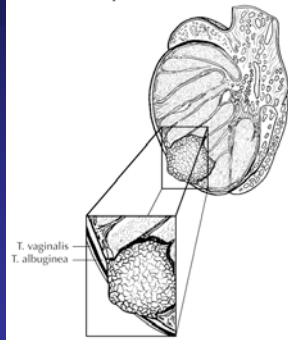
When coding mixed tumors of the testis, it helps to have some guidelines. In the left column of the slide, you see the histologic cell types listed according to prognosis, with the worst being listed first.

If you have a mixed tumor, how do you know what to code? Follow the guideline on the right column. If choriocarcinoma is included in the mix, you would code to 9101 (choriocarcinoma plus other germ cell elements), because that cell type is associated with the worst prognosis. 9081 is a mix of teratoma and embryonal cell carcinomas. If seminoma is mixed with non-seminomas, 9085 is the code. If the mix of cells listed does not include any seminoma, the code is 9065.

Advanced Abstracting Testicular Cancer

II. STAGING

AJCC Cancer Staging Atlas,
2006. Used with permission.



pT2 – CS Extension code 20



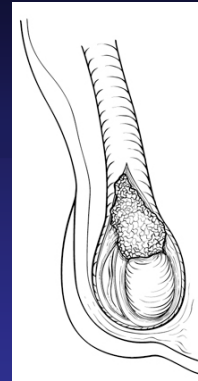
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Now that we have had an introduction to testicular cancer and how it is described and diagnosed, we can further discuss the staging of in the AJCC TNM system and Collaborative Staging.

First, TNM. Testicular is the only chapter in the AJCC book that does not have a group stage IV. Even with distant mets, the highest group stage is III.

AJCC: T Category

- ◆ No clinical T (code 88)
 - pTx if no radical orchiectomy
- ◆ T0 No evidence
- ◆ Tis In situ
- ◆ T1 Limited to testis without vascular/lymphatic invasion
 - Tunica albuginea
- ◆ T2 Limited to testis WITH vascular/lymphatic invasion OR tunica vaginalis
- ◆ T3 Invades spermatic cord
- ◆ T4 Invades scrotum



pT3 Spermatic cord involvement

AJCC Cancer Staging Atlas, 2006. Used with permission.



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Because almost all patients undergo an orchiectomy (which is a pathologic resection), there is no clinical “T” category noted in the chapter. If your computer needs an answer for that field, it could be 88 for not applicable. If an orchiectomy was not done and you are answering the other pathologic categories (lymph nodes, mets), the chapter tells us to use TX. However, many software packages use the pT information from the orchiectomy to group stage the case similar to the collaborative stage rules. Since they take their guidelines from the standard-setters, it is possible this has been approved for this chapter alone. It is recommended that you answer the individual T, N, M with definitions from the book and, if software allows group staging, accept it for study purposes.

T0 would be used when there is no pathologic evidence of cancer in the orchiectomy specimen, but frequently a scar is seen where cancer probably was located.

Tis is possible for in situ tumors, though rare.

T1 is limited to the testicle but no vascular and no lymphatic invasion documented. The tumor may involve the tunica albuginea (inner surrounding layer) but not the tunica vaginalis (outer serosa-like layer).

T2 is limited to the testicle with vascular, or lymphatic invasion documented by the pathologist, or tumor that involves the tunica vaginalis.

T3 tumors involve the spermatic cord. At this point, vascular or lymphatic invasion doesn't matter.

T4 tumors invade the scrotum, and again, vascular or lymphatic invasion doesn't matter.

Regional Lymph Nodes

◆ Abdominal LN

- Interaortocaval
- Para- or peri-aortic
- Paracaval
- Preaortic
- Precaval
- Retroaortic
- Retrocaval
- Retroperitoneal
- Spermatic vein

◆ Other LN ONLY if history of scrotal or inguinal surgery

- Intrapelvic
- External iliac
- Inguinal

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Remember that in the fetus, the testicles developed near the kidneys. Therefore, the regional lymph nodes that formed a connection or lymphatic drainage with the testicles are abdominal lymph nodes—even though they are not the closest lymph nodes to the testicles. Also, laterality doesn't change the code.

However, if the patient had scrotal or inguinal surgery, the lymphatic drainage may have been disrupted to the abdominal area lymph nodes. In that case, the testicular lymph may drain to the inguinal or iliac lymph nodes, and those nodes have now become regional.

How will you know? You need to read the history of the patient carefully. Did he have an inguinal or scrotal hernia surgery in the past, even as a child? How about a cystocele? Was there ever a football injury or car accident causing injury to the groin that was repaired? If there was no history of these events, the inguinal or iliac nodes would be coded as distant metastases.

AJCC: N Category

Clinical

- ◆ Nx Not assessed
- ◆ N0 Not involved
- ◆ N1 LN mass ≤ 2 cm
OR multiple LNs,
none > 2 cm
- ◆ N2 LN mass or multi
LNs > 2 cm but
 ≤ 5 cm
- ◆ N3 LN mass > 5 cm

Pathologic

- ◆ pNx Not assessed
- ◆ pN0 Not involved
- ◆ pN1 LN mass ≤ 2 cm
AND ≤ 5 pos LN
(none > 2 cm)
- ◆ pN2 LN mass > 2 cm
but ≤ 5 cm OR
 > 5 pos LN
(none > 5 cm)
OR extranodal
extension
- ◆ pN3 LN mass > 5 cm

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Coding lymph nodes is similar for both clinical and pathologic classifications. We look at size of the lymph node or the size of the mass. But pathologic N can also include the number of positive lymph nodes, as well as the presence of extranodal extension in pN2.

AJCC: M Category

- ◆ Mx Not assessed
- ◆ M0 Not involved
- ◆ M1 Distant metastases
 - M1a Non-regional node OR pulmonary metastases
 - M1b Distant metastases other than M1a

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M1 is subdivided into two groups:

M1a—non-regional lymph nodes, which could include inguinal or iliac if there has no history of surgery, and/or pulmonary metastases (the most common M1 site for testicular cancer)

M1b—any other site of distant metastasis

Pathologic M0

- ◆ Not possible without autopsy
- ◆ I&R Question #20858
 - AJCC Instructions, page 5
- ◆ What do we do?
 - pT_ pN_ pM [blank] (leave blank but use cM to answer stage group)
 - Don't change old data
 - Physician staging? Education may be needed

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Coding the pathologic M has been constrained by software systems and requirements to fill in all cTNM or all pTNM to arrive at stage. In discussion with AJCC physicians, it was noted that there is no such thing as pathologic M0 for no metastasis. That would require biopsy of every possible metastatic site within the body. Even if a biopsy of one suspected site was negative (such as liver biopsy), it doesn't mean there couldn't be a sanctuary site that had metastases but was not biopsied.

The instructions in the front of the TNM book tell us we can use clinical M information when staging pathologic cases. The problem is that we have linear field groups within our computer software. That is, we answer all the pathologic fields (T, N, M) or the clinical fields (T, N, M), but there is not a place to mix and match the two. We cannot enter pMx, because that would be unknown and cause a stage group of "99". Of course, pM1 means that a distant metastasis was found. So, it is recommended that we leave the pM area blank, but use the cM information to complete the stage group.

If you have not been doing this, what should you do with your old data? Nothing. This was not considered an error by EDITS in the past so it doesn't require a change. Most of us make changes in procedures for the sake of consistency when we begin abstracting a new year. If physicians have been checking the pM box, it may require some education. Hopefully, in the 7th edition of AJCC staging, this may be better explained.

Serum Marker Frequency

| TYPE | Freq % | AFP % | HCG % |
|------------------------|---------------|--------------|--------------|
| Germ cell | 100 | 50-75 | 40-60 |
| Seminoma | 42 | 0 | 9 |
| Non-semin germ | 58 | 65 | 56 |
| Embryonal | 26 | 70 | 60 |
| Teratocarcinoma | 26 | 64 | 57 |
| Teratoma | 5 | 37 | 25 |
| Choriocarcinoma | 1 | 0 | 100 |
| Yolk sac | < 1 | 75 | 25 |

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This table shows how frequently the tumor markers are ordered, and the percentage of time they are secreted by the tumor, according to what cell type is present in the patient's testicle.

Serum tumor markers are part of TNM staging (the S category), and are needed to select the appropriate stage group. They also have other uses.

The S category is based upon combinations of these markers and LDH. See the next slide.

Tumor Markers

- ◆ AFP, hCG, LDH
- ◆ Not used for screening
- ◆ Timing and use
 - Workup: determine cell type
 - Workup: prognosis
 - Postop: residual disease/adjuvant tx
 - Postop: FU/recurrence
- ◆ Issues
 - Availability of information
 - When to document for staging

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The three serum tumor markers associated with testicular cancer are alpha-fetoprotein (AFP), human chorionic gonadotropin (hCG), and lactate dehydrogenase (LDH). They are not unique to testicular cancer, so they are not useful for screening patients. As we saw on the previous slide, not all types of testicular cancer secrete markers.

These markers can be used for different purposes throughout the course of the patient's disease. When the physician is suspicious of the diagnosis, the markers can indicate what type of cancer is expected if they are abnormal. Also, the higher the marker, the higher the suspicion of extensive disease involvement, perhaps guiding the physician to request more radiologic workup for distant mets.

Postoperatively, the markers should be followed sequentially immediately following orchiectomy and within 30 days to look for the level of decrease from a positive or abnormal level. If the marker does not fall quickly enough, it could indicate that the pathologist missed some non-seminatous germ cell element in the specimen, suggesting adjuvant chemotherapy may be required.

Finally, they can be used to monitor the patient for recurrence of the tumor. Again, only if they were positive preoperatively.

There are issues with these markers. The odds are that the lab work for the markers was done at, or soon after, the appointment with the physician and may not have been done in your facility, but at an outpatient lab. Therefore, the registrar may not have the information available from preoperative lab work. Not all three markers are usually ordered. LDH is actually quite a nebulous marker, and is probably ordered less than half the time, primarily if there is strong suspicion that the patient may have metastatic disease. In AJCC staging, all three are required for complete stage grouping, so how do the doctors stage the patient when there are only two available? Usually they use AFP and HCG, and do not worry about the lack of information from LDH.

Lastly, when should these markers be documented? In a community hospital setting, you may have the information of the pre-admit markers documented in the H&P prior to the orchiectomy. The AJCC chapter states the markers should be documented following surgery, and that information is usually not available to the hospital registrar, since it will be done back in the physician's office. This also makes the documentation of these markers inconsistent with the markers we document for other cancers. Furthermore, there is a strong likelihood that they are not being recorded consistently by registrars. If the markers are to be used for prognosis, that would imply pre-operative results should be gathered. If they are necessary for adjuvant treatment decisions, then postoperative results would be more important. AJCC has been asked for clarification about this issue, but the answers may wait until the 7th edition is published.

We will talk more about how to calculate and code them for collaborative staging in later slides.

CS Tumor Size

- ◆ Use Standard Table

- Size of cancer, not testicle

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Let's look at coding Collaborative Stage.

Tumor size is not really a major piece of information to determine stage. If the information is available, we record the size of the cancer, not the size of the testicle.

CS Extension

- ◆ No clinical T categories
- ◆ Except Tis and T4, need radical orchiectomy (SSF4) and markers
- ◆ 10 Invasive WITHOUT vascular/lymph invasion or NOS
- ◆ 15 Invasive WITH vascular/lymph
- ◆ 20 Tunica vaginalis, surface implants

10, 15 include

- Body of testis
- Rete testis
- Tunica albuginea

28



In order to derive a stage group in TNM, the T1–3 values are based on whether an orchiectomy was performed, which is why we have Site-specific Factor 4.

In general, codes 10–20 would be local tumors, probably T1 category, if markers are low. Codes 10 and 15 include the body of the testis, the rete testis, and the tunica albuginea.

CS Extension

- | | |
|---|---|
| <p>◆ 40 Epididymis WITHOUT vascular/ lymphatic invas or NOS</p> <p>◆ 45 Epididymis WITH vascular/ lymphatic invas</p> <p>◆ 50 Spermatic cord, ipsilateral OR vas deferens</p> | <p>◆ 60 Dartos muscle, OR scrotum, ipsilateral</p> <p>◆ 70 Extension to scrotum, contra- lateral OR ulcerated scrotum</p> <p>◆ 75 Penis</p> <p>◆ 80 Further extension</p> <p>◆ 95 No evidence</p> |
|---|---|

29



Codes in the left column derive Regional by Direct Extension tumors in SS 2000. AJCC T1 to T3 derivation depends on marker results and lymphatic or vascular invasion in the primary tumor.

Codes in the right column indicate T4 tumors. Codes 70–80 are distant in summary staging. The dartos muscle is the only muscle within the wall of the scrotum, and the job of this muscle is to regulate heat. If the body is cold, the muscle contracts and causes the scrotum to get wrinkled, thereby creating less surface area to let heat out. Temperature is very important for spermatogenesis. The dartos muscle works with the cremaster, muscle which helps move the testicles up and down inside the scrotum.

Resources: Collaborative Stage Manual, Gray's Anatomy

CS Lymph Nodes

- ◆ Needs number of pos LNs and SSF5
- ◆ Bilateral OR contralateral
- ◆ 00 None
- ◆ 10 Aortic, retroperitoneal, spermatic vein, regional, NOS
- ◆ 20 Pericaval
- ◆ 30 Pelvic NOS, external iliac
- ◆ 40 Inguinal
 - Deep, superficial
 - Node of Cloquet or Rosenmüller
- ◆ **30 or 40 WITH previous scrotal or inguinal surgery**
- ◆ Bilateral OR contralateral

30



In order to generate an AJCC N value, we need to know the number of positive LNs, as well as the size of the metastasis from Site-specific Factor 5.

For codes 10 to 30, it doesn't matter if the nodes are ipsilateral, contralateral, or bilateral.

Codes 30 or 40 require documentation of previous scrotal or inguinal surgery. If there has been none, these positive LNs are coded as distant metastases in the Mets at Dx field.

Question: If pelvic nodes are considered regional because of disruption of lymphatic channels, do abdominal nodes remain regional as well, or do they become distant lymph nodes?

Answer: Scrotal surgery *adds* inguinal lymph nodes as additional regional drainage, but the abdominal/retroperitoneal still remain regional as the primary route of spread. The inguinal nodes are basically collateral drainage, and don't become the primary route.

CS Mets at Dx

- ◆ 11 Pelvic, external iliac LN
- ◆ 12 Inguinal LN
 - 11 or 12 - *WITHOUT previous scrotal or inguinal surgery*
- ◆ 13 Distant LN not in 11 or 12
- ◆ 20 Mets to lung
- ◆ 25 Mets to lung AND LNs
- ◆ 40 Mets to OTHER sites (with/without lung or LNs); carcinomatosis
- ◆ 45 Distant mets, NOS

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Codes 11–25 derive M1a. Codes 11 and 12 include distant LNs that might be regional, but no previous surgery to area was documented. All other distant LNs (supraclavicular, axillary, etc.) are 13.

Code 40 is M1b, Code 45 is M1, NOS.

CS Evaluation Fields

◆ Use Standard Table

- WHERE did info come from?
- NOT was something done?

Example: Patient had radical orchiectomy for seminoma. CT scan abdomen showed retroperitoneal adenopathy. PET scan confirmed adenopathy but no other mets seen.

T Eval: 3 (resection) LN Eval: 0 (CT/PET)

Mets Eval: 0 (CT/PET)

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Coding Evaluation Fields for CS is frequently misunderstood. We need to go back and read the front of the CS Manual from page I–30 on. We are supposed to use the Eval Field to describe how we came by the information coded in the field. That means all of the pieces of information and especially the piece of information that described the greatest extent or code hierarchy.

In the example, there was a resection of the primary tumor done, but that only helps us answer the tumor size and extension fields. The answers for lymph nodes and metastasis came from clinical information, namely the CT and PET scans. So we're coding each field with a "how do I know about that" type of question/answer. How do I know about the tumor invasion? From a radical orchiectomy (Eval code 3). How do I know about the LN status? From a CT scan (Eval code 0). The radical orchiectomy would not remove regional nodes, so we can't use code 3 in Nodes Eval. How did I rule out distant mets? From the CT scan (Eval code 0).

CS Site-specific Factor 1 – AFP

CODES

| | | |
|-----|------------------------------|--|
| 000 | Not done | ◆ Not produced by pure seminomas or choriocarcinomas |
| 020 | Within normal limits (S0) | |
| 040 | Range 1 (<1,000 ng/ml) | ◆ Half-life: 5–7 days |
| 050 | Range 2 (1,000–10,000 ng/ml) | ◆ If elevated with “pure” seminoma, patient should be treated as if non-seminoma |
| 060 | Range 3 > 10,000 | |
| 080 | Ordered, results? | |
| 999 | Unknown, no info | |

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Alpha-fetoprotein (AFP) is the first of three serum tumor markers coded in site-specific factors. Alpha-fetoprotein should be less than 25 ng/ml in the normal male. However, increased levels of AFP are also found in patients with other malignancies (pancreas, biliary, gastric, and lung); as well as liver diseases such as cirrhosis, acute and (chronic) hepatitis, and hepatic necrosis.

What is a half-life? Within the time listed, the original level should be half of what it was. For example, let's say the AFP was 1,000 at diagnosis. Within the 7 days noted, a second AFP test should show a level of < 500. Within the next 7 days, the level should be 250. And 7 days after that, it should have fallen below 125. That's three weeks later. Knowing how high it was to start with would help the clinician determine the result he should see a month later, if the orchiectomy alone was influencing this marker.

If the marker is elevated beyond what the half-life calculation shows, it is possible the pathologist missed some non-seminomatous element in the specimen, and the patient should be considered for adjuvant treatment. Or, if the marker is elevated and the CT scan was read as possible adenopathy, it is possible that the marker is indicating those lymph nodes are positive and adjuvant treatment should be done. Doctors do not consider this recurrence, but a definition of where all the disease is. We will talk about lymph node dissection in a few slides, and you will see that this is not an option that doctors are anxious to perform if not required.

This is one cancer where it takes time to decide if adjuvant treatment should be offered based of performance of these markers.

Resources: www.aafp.org, www.tc-cancer.com, American Cancer Society, Collaborative Stage Manual

CS Site-specific Factor 2 – hCG

CODES

| | | |
|-----|------------------------------|---|
| 000 | Not done | ◆ Not detectable in healthy males |
| 020 | Within normal limits (S0) | ◆ Half-life: 16–24 hours |
| 040 | Range 1 (<5,000 ng/ml) | ◆ 90% decrease every 21 days should be noted during chemo |
| 050 | Range 2 (5,000–50,000 ng/ml) | ● If not, residual tumor? Drug resistance? |
| 060 | Range 3 > 50,000 | |
| 080 | Ordered, results? | |
| 999 | Unknown, no info | |

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The second tumor marker is Human Chorionic Gonadotropin, although it was the first to be noted to have a relationship with testicular cancer in the 1930s. Yes, this is the same hCG that is used in women to indicate pregnancy or gestational disorders. It is also elevated in other malignancies (liver, biliary tract, pancreas, stomach, lung, breast, kidney, and bladder).

hCG level can become elevated (falsely positive) due to abnormally low levels of testosterone or because of marijuana use.

When looking at the half-life, if it's not decreasing appropriately, the physician should discuss concerns with possible residual disease (for example, should the patient have a lymphadenectomy?) or drug resistance to the chemotherapy chosen.

Resources: www.aafp.org, www.tc-cancer.com, American Cancer Society, Collaborative Stage Manual

CS Site-specific Factor 3 – LDH

CODES

| | | |
|-----|---------------------------|---------------------------------------|
| 000 | Not done | ◆ Elevated in 50% patients |
| 020 | Within normal limits (S0) | ◆ Not particular to tumor type |
| 040 | Range 1 (1.5 x N) | ◆ May not be done |
| 050 | Range 2 (1.5 – 10 x N) | |
| 060 | Range 3 > 10 x N | ◆ Example: Common range: 105-333 IU/L |
| 080 | Ordered, results? | ● 1.5 x 333 = 499.5 |
| 999 | Unknown, no info | |

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The last marker is Lactate Dehydrogenase. What is “N”? It is the upper limit of normal for this test in your lab. Clinically, it is useful as a marker of advanced or bulky disease and, when elevated, as a marker for seminoma. However, it may not be done because the function of the LDH test is to help identify the cause and location of tissue damage in the body, and that’s pretty non-specific. LDH can be run for a multitude of diseases or conditions.

How do you calculate whether the LDH is in range 1, 2, or 3? Multiply the upper limit of normal by 1.5.

In the example, if the patient’s LDH was 400, it would be within Range 1 because that is less than 499.5 (1.5 times the upper limit of 333). But if the patient’s LDH was 600, it would be in Range 2 (more than 1.5 times the upper limit but less than 10 times the upper limit). In this case, Range 2 would encompass values from 499.5 to 3,330.

Resources: www.aafp.org, www.tc-cancer.com, American Cancer Society, Collaborative Stage Manual

CS SSF 4 – Radical Orchiectomy

- ◆ 000 Not performed
 - ◆ 001 Performed
 - ◆ 999 Unknown if performed
-
- ◆ Used to determine if T should be pathologic.

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Site specific factor 4 is pretty simple to code. Was the orchiectomy performed? It's a yes, no, or unknown answer.

By answering this factor, the CS algorithm can determine if the T output should be clinical or pathological information.

CS SSF 5 – Size of Mets in LN

CLINICAL

001 ≤ 2 cm (N1)

002 > 2 but ≤ 5 cm (N2)

003 > 5 cm (N3)

PATHOLOGIC

001 ≤ 2 cm AND no
extranodal
extension

002 > 2 but ≤ 5 cm OR
extranodal
extension

003 > 5 cm N3

998 NOS, size,
number, or
extranodal not
stated

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The left column shows us how we would code lymph node size based only on clinical assessment (CT scan, MRI, etc.). We should review the estimate of the lymph node mass on the radiology report and/or review the TNM from the physician to choose the code for this field.

The right column shows us what would be documented in the path report in order for us to determine the code.

There is no SSF 6 in the testicular scheme (use code 888 for not applicable).



Testis is one tumor site that requires some follow-up of patients before you can decide if first course of therapy is completed.

Example: Patient has orchiectomy. Then you must wait several weeks to see if tumor markers become totally negative. If they do not, there are several questions to ask:

Was the path report correct, or was some other type of histology missed?

Did markers never get negative or start to rise? If so, patient needs some more treatment.

If CT scan showed negative lymph nodes but markers are abnormal, is imaging correct, or could there be disease there?

If possible lymph node disease, does patient need RT, versus observation, versus LN dissection?

If patient has RT to nodes or observation, does marker stay negative, or does patient still need dissection?

In most other sites, the adjuvant treatment can be decided based on the path report from the dissection. Evaluation of testicular cancer takes more time than that, so don't get anxious and think the patient has recurrence when the reality is that the first course has just not been finished. If you are abstracting the case within the 4–6 month time frame, the physician should have had enough time to gather several markers to make the decision about adjuvant therapy.

Surgery

◆ Orchiectomy

- Simple
- Subcapsular
- **Radical or inguinal for testicular CA**
- Why inguinal surgery?
 - ◆ Fear of spreading cancer via scrotal skin
- Why not just chemotherapy?
 - ◆ Chemotherapy drugs not taken in by testes

For gender reassignment
or prostate cancer

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There are multiple types of orchiectomies. The first two (simple and subcapsular) are not usually done for testicular cancer.

A radical orchiectomy is done via an incision in the groin (where the thigh meets the torso). It is done through the groin, so the cancer cells do not have the chance to contaminate the scrotal skin and move to the other testicle or penis.

Why not just treat the patient with chemotherapy, since this disease is so responsive? Chemotherapy does not get into the testicle to work on the cancer cells there.

FORDS Surgery Codes

- ◆ 12 Local destruction NOS (no path)
- ◆ 20 Local/partial excision
- ◆ 30 Orchiectomy WITHOUT spermatic cord
- ◆ 40 Orchiectomy WITH cord or cord NOS
- ◆ 80 Orchiectomy, NOS
- ◆ 90 Surgery, NOS



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Coding surgical procedures is pretty simple. Since the overwhelming majority of patients will have an orchiectomy, your code choice will most likely be between 30 and 40. Discourage use of code 80 for analytic patients.

The spermatic cord includes the lymph and blood vessels, vas deferens, and other structures that suspend the testis in the scrotum. Removal of the spermatic cord allows the pathologist to better classify the T category.

Other Surgical Issues

- ◆ **Sperm banking**

- ◆ **Implants**

- **Silicone**
- **Saline**



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Sperm banking is also an issue that should be discussed with a young male patient. This is done after the orchiectomy but prior to initiation of chemotherapy or radiation. The sperm are frozen (cryopreserved) until the day the patient decides that he is interested in fathering a child.

It is possible that the patient will consider having a testicular implant. The Testicular Cancer Resource Center conducted an informal comment poll of patients a few years ago and found that most recommended against it. It may be difficult to find physicians who offer this procedure in the USA, although we found doctors on the web from Canada, Argentina, and France. As with women and breast implants, there are two different materials, which the FDA regulates. Silicone implants are said to be firmer, saline implants are said to be softer, but more expensive.

Retroperitoneal Lymph Node Dissection

YES

- ◆ Stage II or higher nonseminoma (possibly Stage I)
- ◆ If tumor markers do not decrease enough

NO

- ◆ Seminomas
- ◆ Rising marker after orchiectomy
- ◆ If RLND can't be done within 1 month
- ◆ Non-USA patients

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Retroperitoneal lymph node dissection—to do or not to do? It is usually recommended in nonseminomas unless the lymph node mass is very large. It may also be recommended if the patient has an orchiectomy but the tumor markers do not decrease fast enough, indicating that there are lymph nodes that are positive.

It is NOT done for pure seminomas. If the marker rises after orchiectomy, the patient should go right to chemotherapy, rather than take time to recover from this major surgery and wait on starting chemotherapy. A lymph node dissection should be done within a month of the orchiectomy. If it's much longer than that, chemotherapy should be initiated. And why not for non-USA patients? In Europe, there are not enough urologists with experience doing retroperitoneal lymph node dissections, and chemotherapy can be almost as effective.

If retroperitoneal lymph node dissection is done as an open procedure, the incision runs from just below the sternum to around the belly button. The procedure can take four hours or more. Multiple complications are associated with this surgery including retrograde ejaculation (due to nerves being cut, the sperm ends up in the bladder instead of the normal route). In recent years, there is more effort going toward laparoscopic dissection.

What other choices are there? The patient could choose radiation to the retroperitoneum. Or the patient could choose close observation in certain circumstances. We'll look at the choice of observation in just a minute.

Resource: <http://tcrc.acor.org>, <http://www.tc-cancer.com>

Lymph Node Radiation

- ◆ External beam
- ◆ Instead of RLND
- ◆ Seminomas more sensitive
- ◆ Dose 25-35 Gy
- ◆ Side effects

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A patient may have the option of radiation to regional lymph nodes rather than a surgical dissection for Stage I and Stage II disease, especially if the diagnosis was a seminoma, which is more sensitive to radiation.

Usually the treatment will be 25 Gy over a few weeks with a possibility of 10 more Gy if patient had Stage II disease. What are the side effects of radiation? Fatigue, diarrhea, nausea and interference with sperm production—all of which may be temporary.

There was a study that showed one course of chemotherapy would be just as effective as radiation to lymph nodes in Stage I disease. However, long term follow-up has not been completed or reported, and that may change the usefulness of this idea.

Surveillance

Nonseminoma

- ◆ Year 1–Tumor markers and CXR every month; CT abdomen every 2 mo.
- ◆ Year 2–Markers and CXR every 2 mo.; CT abdomen every 4 mo.
- ◆ Years 3–5 –Markers and CXR every 6 mo.; CT abdomen every 6 months
- ◆ After Year 5–Markers and CXR yearly

Seminoma

- ◆ Year 1–umor markers and CXR every 2 months; CT abdomen every 3 mo.
- ◆ Year 2–Markers and CXR every 2 mo.; CT abdomen every 4 mo.
- ◆ Years 3–5 –Markers and CXR every 6 mo.; CT abdomen every 6 months
- ◆ After Year 5–Markers and CXR yearly

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Source: www.acor.org/tcrc



According to the Testicular Cancer Resource Center website, 70% of men diagnosed with Stage I nonseminoma and 80% of men diagnosed with Stage I Seminoma are cured by orchiectomy alone. How do we know this? By surveillance.

What does surveillance entail? That depends on the cell type. With proven nonseminomas, there will be closer surveillance for fear that choriocarcinoma may have been one of the elements that could have been missed. With any abnormality in surveillance, the patient could be started on chemotherapy immediately, or opt for lymph node dissection.

With seminoma, follow up is just as frequent in the Oregon Health and Science University protocol shown on the slide. There is another protocol from Princess Margaret Hospital that is a little less stringent with markers being done every 4 months, and radiology every 8 months for the first few years. The concern with seminoma is that there may have been a nonseminomatous element within the path specimen that was missed, so close follow-up would allow the patient to be put on chemotherapy or have a lymph node dissection sooner.

Surveillance

Yes to Surveillance

- ◆ Avoid RLND or chemo
- ◆ Not good time for more treatment
- ◆ Keep appointments
- ◆ Safety net

No to Surveillance

- ◆ “Doing nothing” worry
- ◆ Odds recurrence high
- ◆ Tumor markers unreliable
- ◆ Can’t keep appointments
- ◆ Get treatment over with

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What might guide the patient in making this decision if the physician gives him the option?

The patient may choose surveillance if he wants to avoid the node dissection or chemotherapy for many reasons such as fear, finances, and so forth. The patient may have career or social obligations that mean he doesn't want to take up to three months to recover from more treatment right now. The patient may wish to father a child using the natural method. The patient has to promise to keep the appointments with the doctor, as well as go in for the labwork and radiology as required. That's an interesting conundrum because males do not usually seek medical care without someone motivating them. You may see a patient initiate surveillance but within a year, he's not able to maintain the close contact it requires with the oncologist, and the plan falls apart. At least there is a safety net in surveillance, in that treatment can be started at a later date with very good results in cure and survival.

Why would a patient refuse surveillance? Some cannot get over the worry that doing nothing is allowing the cancer to flourish. Some may have higher recurrence odds with markers or vascular invasion that would make the surveillance less certain. Some patients do not have elevated tumor markers at diagnosis, which would make that an unreliable source of information about their cancer after orchiectomy. Some have careers that necessitate frequent travel and keeping appointments is not possible. And some may just want to get the treatment over with so they can go back to a normal life.

Resource: <http://tcrc.acor.org>

Chemotherapy

- ◆ BEP (Bleomycin, Etoposide, Cisplatin)
- ◆ VIP (Etoposide, Ifosfamide, Cisplatin)
- ◆ VeIP (Vinblastin, Ifosfamide, Cisplatin)
- ◆ Especially Stage II, Stage III



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Back in 1970, about 90% of men with testicular cancer died of their disease. And then cisplatin was shown to be effective for testicular cancers. There is one thing to consider about chemotherapy. Why does it work so well on testicular cancer? Because those cells have a high doubling time. Testicular cancer cells double every 30 days (as compared to breast cells doubling every 60 days, and colon cells doubling every 90 days). That means that cells are more often preparing to divide, which is when the cell is most vulnerable to damage from chemotherapy.

Men with stage II disease should consider chemotherapy, although they may also be considering lymph node dissection versus radiation. Men with Stage III disease need chemotherapy. Patients with Stage I nonseminoma with abnormal tumor markers that have not dropped to normal (Stage IS) **must** be treated with chemotherapy.

The number of courses depends on the type of cancer, the stage, whether lymph node dissection was done, and how markers responded—lots of variables. The patient wants the fewest number of courses that are curative due to side effects. Bleomycin can cause lung fibrosis. Etoposide or VP16 can lead to subsequent leukemia, and Cisplatin can cause neurotoxicity and hearing damage.

There is not much going on with Clinical trials. Some work had been done in the past with stem-cell transplant, but nothing has been reported lately.

Source: <http://tcrc.acor.org>

Treatment Overview: Seminoma

| | |
|-----------------------|--|
| Stage I | RT to retroperitoneal and ipsilateral inguinal nodes |
| Stage IIA | RT to retroperitoneal and inguinal nodes, possibly with mediastinal and supraclavicular nodes |
| Stage IIB, IIC | Platinum-based combination chemotherapy or RT, as in IIA |
| Stage III | Platinum-based combination chemotherapy, possibly with resection of residual mass |

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This slide is a quick snapshot of what you might need to look for when completing an abstract on seminomatous testicular cancer. Each of these treatment plans assumes that the patient has had a radical orchiectomy.

Treatment Overview: Nonseminoma

| | |
|-----------------------|--|
| Stage I | RLND or surveillance |
| Stage IIA | RLND, possibly with platinum-based combination chemotherapy |
| Stage IIB, IIC | Platinum-based combination chemotherapy, followed by resection |
| Stage III | Platinum-based combination chemotherapy, possibly with resection of residual mass |

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This slide is a quick snapshot of what you might need to look for when completing an abstract on nonseminomatous testicular cancer. Again, the treatment plans assume that a radical orchiectomy has been done.

Salvage Treatment

- ◆ Original treatment
- ◆ Type of recurrence
- ◆ Chemotherapy
- ◆ Surgery
- ◆ Stem cell
- ◆ Clinical trial

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The choices for salvage treatment depend on how the patient was treated originally as well as what cell type is actually relapsing, especially if the patient had a mixed tumor. What type of recurrence (Lymph nodes? Distant organ?) as well as what cell type can play a role in the treatment choices for salvage. Biopsy of the recurrence or close monitoring of the markers may help illustrate which cells have returned. Is only the teratoma seen? Or are there embryonal cells?

If no chemotherapy was done in first course, that could be an option. If chemo was done, choosing a different regimen may still give an 80% response rate.

Single metastases found in another organ or lymph node metastases may be effectively resected, followed by chemo with good results, especially if the recurrence happened two years or more after initial treatment.

This is an area where researchers once thought bone marrow transplant would work, but comparison trials showed transplants had much more toxicity and the transplant did not lead to any survival advantage. However, there may be clinical trials available for the relapsed patient and these should be discussed.

If a patient with a mixed cell testicular cancer later develops a cancer consisting of only one of the previous cell types, the new disease is recurrence of the original cancer, not a new primary. The cancer has *transformed* due to the treatment given to the original cancer. It is possible that the remaining cell type is resistant to what was given in first course of treatment, and additional therapy for the recurrence/transformation is needed.

Advanced Abstracting Testicular Cancer

IV. OUTCOMES Follow-up and Survival

| | | | |
|---|---|---|---|
| <small>Yr</small> 1970 1971 1972 1973 1974 1975 1976 1977 1978 1979 1980 1981 1982 1983 1984 1985 1986 1987 1988 1989 1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022 2023 2024 2025 2026 2027 2028 2029 2030 | <small>Yr</small> 1970 1971 1972 1973 1974 1975 1976 1977 1978 1979 1980 1981 1982 1983 1984 1985 1986 1987 1988 1989 1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022 2023 2024 2025 2026 2027 2028 2029 2030 | <small>Yr</small> 1970 1971 1972 1973 1974 1975 1976 1977 1978 1979 1980 1981 1982 1983 1984 1985 1986 1987 1988 1989 1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022 2023 2024 2025 2026 2027 2028 2029 2030 | <small>Yr</small> 1970 1971 1972 1973 1974 1975 1976 1977 1978 1979 1980 1981 1982 1983 1984 1985 1986 1987 1988 1989 1990 1991 1992 1993 1994 1995 1996 1997 1998 1999 2000 2001 2002 2003 2004 2005 2006 2007 2008 2009 2010 2011 2012 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022 2023 2024 2025 2026 2027 2028 2029 2030 |
| | ★ | | ★ |
| | | ★ | ★ |
| ★ | | ★ | |

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5-Year Survival

| Stage | Seminoma | Non-Seminoma | Overall |
|------------|----------|--------------|---------|
| Stage I | 99% | 98% | 98% |
| Stage II | 95% | 95% | 95% |
| Stage III | 90% | 76% | 78% |
| All Stages | | | 96% |

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Because so many patients are found at early stage, and because the disease responds so well to chemo and radiation, the survival is one of the best in all of the cancers we track.

Reference: <http://tcrc.acor.org/>

Follow-up

- ◆ Most recur within first 3 years
- ◆ Every 2–3 months for Year 1
 - Then every 3–6 months Year 2
 - Then every 6 months until Year 5
- ◆ Should include PE, lab markers, CXR, CT abdomen

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Even if a patient did not choose surveillance, he should be closely monitored as documented on the slide. The monitoring is for multiple purposes. Recurrence of the original cancer, a new cancer in the other testicle, a new cancer as a side effect of treatment (especially chemo), or just a new cancer because the immune system has proven to be faulty.

And with this, our review of testicular cancer is completed. Thank you for your attention.

Resources

- ◆ Association of Cancer Online Resources (www.acor.org/tcrc)
- ◆ National Cancer Institute (www.cancer.gov)
- ◆ *Comprehensive Textbook of Genitourinary Oncology*. PA: Lippincott, 2000

Additional Resources

Images

- ◆ *AJCC Cancer Staging Illustrations in PowerPoint* from the AJCC Cancer Staging Atlas, sixth edition (2002). Springer-New York, 2007. Used with permission.
- ◆ A.D.A.M. Interactive Anatomy 4, A.D.A.M., Inc., 2004. Used with licensed permission.

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These are some additional websites and programs that provided pictures information for this presentation.

The findings and conclusions in this presentation are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

**For information about CDC's
Cancer Prevention and Control Programs
and the
National Program of Cancer Registries**

Please visit

www.cdc.gov/cancer/npcr

